and dual X-ray absorptiometry findings at 5 years, compared with the placebo group.

These results show that calcium supplementation can reduce the risk of osteoporotic fracture effectively, but only when the tablets are consistently taken. The authors conclude that calcium supplementation is unlikely to be a successful preventative public-health initiative, because long-term compliance cannot be assured.

Original article Prince RL *et al.* (2006) Effects of calcium supplementation on clinical fracture and bone structure: results of a 5-year, double-blind, placebo-controlled trial in elderly women. *Arch Intern Med* **166:** 869–875

Adiponectin and insulin resistance associated with endometrial cancer

A study by Soliman *et al.* reports that low levels of adiponectin are a risk factor for endometrial cancer. Levels of this protein, which is secreted by adipose cells, show a negative correlation with the prediabetic state of insulin resistance and hyperinsulinemia; these conditions might therefore contribute to endometrial cancer development.

Serum adiponectin levels were measured in 117 women with endometrial cancer (cases) and 238 women with no cancer history (controls), and their association with endometrial cancer determined. Mean serum adiponectin levels were significantly lower in cases than in controls (88.8 ± 63.3 ng/ml and 148.2 ± 68.3 ng/ml, respectively; $P<0.001$), a difference that remained after adjustment for age, BMI, diabetes, and hypertension. As expected from previous studies, age, BMI and hypertension were all independently associated with endometrial cancer, but serum adiponectin level showed the strongest association, with cases over 10 times more likely to have adiponectin levels in the lowest tertile, and nearly 3 times more likely to have adiponectin levels in the intermediate tertile, than controls ($P<0.001$ and $P=0.05$, respectively). Obesity is widely regarded as the strongest risk factor for endometrial cancer; this study showed a strong association between adiponectin level and endometrial cancer even in the subset of non-overweight (BMI <25 kg/m^2) women, and indicates that insulin resistance is independently associated with this cancer.

Soliman *et al.* comment that data concerning other potential risk factors in their patients were lacking, and state that their results need confirmation in a prospective longitudinal study that calculates the relative risk associated with insulin resistance and developing endometrial cancer.

Original article Soliman PT *et al.* (2006) Association between adiponectin, insulin resistance and endometrial cancer. *Cancer* **106:** 2376–2381

Normal values for growth hormone should be reduced for obese adults

The diagnosis of growth hormone deficiency in adults is always challenging, but it is particularly difficult in the case of obese adults. As obesity is known to independently decrease growth hormone secretion, it is possible that the normal cutoff values for tests commonly used to detect growth hormone deficiency do not apply to obese individuals. Kelestimur *et al.*, therefore, assessed the influence of obesity on GHRH + GHRP-6 (growth-hormone-releasing hormone plus growth-hormone-releasing peptide 6) test results. This test measures the patient's ability to secrete growth hormone.

In total, 542 individuals were tested: 50 obese and 126 nonobese participants with hypopituitarism and growth hormone deficiency, and 105 obese or morbidly obese and 261 nonobese participants with normal pituitary function. At present, a peak value of ≥20 μg/l indicates normal growth hormone secretion, and a peak value of ≤10 μg/l indicates deficiency. Analysis revealed that the existing cutoff values were valid for individuals with a BMI ≤35/kg/m^2, but that the cutoff values for both normality and deficiency should be lowered by 5 μg/l for individuals with a BMI >35/kg/m^2.

These results confirm the influence of obesity on growth hormone secretion, and on GHRH + GHRP-6 test results. Surprisingly, the authors found that obesity itself accounts for only 25% of the reduction in growth hormone secretion observed in obese individuals. As the authors could offer no explanation for this surprising finding, it might be an interesting area for further study.

Original article Kelestimur F *et al.* (2006) Effect of obesity and morbid obesity on the growth hormone (GH) secretion elicited by the combined GHRH + GHRP-6 test. *Clin Endocrinol* **64:** 667–671